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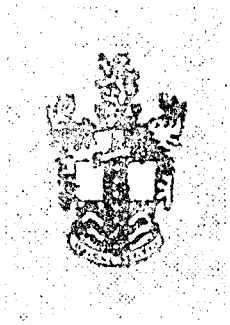

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## AN INTRODUCTION TO THE INFORMATION PROCESSING COMPONENTS OF THE BRAIN

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# An Introduction to the Information Processing Components of the Brain

Dr. S. Collins

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**Abstract**

Over the past decade there has been increasing interest in neurologically inspired computational techniques. This interest arises from the concurrence of two factors; firstly, a growing list of interesting tasks for which serial digital computers are unsuitable, and secondly, information gained from the application of new techniques in neurobiology. This text is intended to provide an introduction to neurobiological terms for physical scientists and engineers, with some pointers to further reading. As an introduction little prior knowledge is assumed and the text begins with a short description of a generic neuron. This description is followed by more detailed discussions of those aspects of neurobiology of particular importance in information processing. The topics covered include the following. The electrical signals used to represent information. The ion conduction mechanisms employed by cells to support these signals. The behaviour of cell membranes and intercellular junctions. Finally a few comments on the immediate implications of even a superficial understanding of neurobiology are included.

## Contents

# 1 Introduction

This text aims to provide a simple, informative introduction to neurobiology for engineers and physical scientist. The demand for such an introduction has been generated by the current resurgence of interest in *neural networks* among engineers and physical scientists. Although each scientist will have a personal motivation for this interest, the general trend is to examine biological systems in the hope of gaining insight which will help solve particular problems.

This approach is not as revolutionary as it might first seem. There have been previous attempts to study the brain to assist the development of "computers"[1, 2]. The most notable success in this area was the development in 1943, by McCulloch and Pitts, of a thresholding logic unit as a formal model of a neuron. Although abstract, this model has successfully served two functions; first, it formed the first basis for understanding information processing in the brain, second, it has spawned a model capable of universal computation. The perceptron, and hence its generalisation the multi-layer perceptron (MLP), are well known architectures built from these neurons.

There has been considerable progress since 1943 in both neurobiology and technology. The advances in technology have generated both new problems and potential solutions. It is hoped that neurobiology will offer hints as to how to apply the technology to solve particular problems. Our present level of understanding of biological systems suggests that this approach may be productive when applied to such low level tasks as pattern recognition, image processing and associative memories. On the contrary attempts to examine biological systems to identify the basis of intelligence, as the first step to building a "thinking" machine, would appear to be premature.

Attempting to understand the brain is a daunting task. It is common to find statements that the human brain contains $10^{11}$ cells and approximately $10^{15}$ connections [1]. We can only begin to study this staggering number of cells because large numbers of cells can be categorised together. Within a category each cell is slightly different from the others, but, each category is as identifiable as a species of tree.

The task of understanding the functioning of the brain is further simplified by the fact that the cells are organised into *centres*. Each centre is distinct and performs specific roles. The cerebellum is one such centre, which controls movement using various sensory inputs. This centre is an example of the complexity which can emerge from large scale repetition of a small number of *basis features*. The whole cerebellum is made up of only five categories of cell. One category, the Purkinje cells, are the medium for all the output from the centre, with each Purkinje cell receiving input from an estimated $8 \times 10^4$ granule cells. The Purkinje cells also take inputs from basket and stellate cells, these categories being distinguished by their connectivity and position within the centre. The last category of cell, the Golgi cells, are the only cells not to interact with the Purkinje cells, they connect together large numbers of granule cells, before the granule cells connect to the Purkinje cells [3].

It is the organisation of the brain into centres containing a finite number of cell categories which forms the basis for any reductionist attempt to understand the brain. The remainder

[1] These figures are estimates which change from author to author

of this text is organised to reduce the centres to their component cells, the *neurons*, followed by a further reduction of neurons to their constituents.

## 2 Individual Cells

The brain consists of two general classes of cells, the *neurons* and the *glial* cells, with each class accounting for approximately half the brain volume. The function of the glial cells appears to be to support the activites of the neurons. Although the glial cells and neurons interact, the information processing functions, if any, of these interactions are unknown. We will therefore concentrate upon the neurons.

A generic neuron can be described in three parts:

1. An array of fine fibres called *dendrites*. The structure of the array resembles that of the branches of a tree, a similarity which is so strong that this feature is usually described as a *dendritic tree*. Closer examination of the dendrites show that they are covered with small structures, *synapses*, which form the connections between neurons. To communicate, two neurons juxtaposition specialist areas of membrane, these sites are the synapses. At a synapse the two membranes are not sufficiently close to actually touch, however, the two sides do almost enclose the *synaptic cleft*, a pocket of intercellular fluid. Most synapses are positioned on the membrane of a neuron. However, a significant proportion of the synapses in the brain are connected to a dendrite via a branch known as a *dendritic spine*.

2. The dendritic tree converges on the cell body, *soma*. The soma contains the structures which perform the functions required to keep the cell alive. The nutrients and other produces of these functions reach the rest of the neuron by diffusion.

3. Emanating from the soma is a long *axon*. At the root of the axon the soma forms a region known as the *axon hillock*, which plays an important role in amplifying signals to be transmitted down the axon. Some neuron types have more than one axon whilst some axons branch, forming *daughter* axons. In practise the distinguishing between dendrites and axons is not easy[4]. The term axon is therefore usually applied to the easily distinguishable myelinated axons. A myelinated axon is identifiable by its covering of insulation, *myelin*, which is periodically punctuated by gaps known as the *nodes of Ranvier*. Unfortunately the existence of unmyelinated axons means that the identification of an axon simply by the presence of myelin is ambiguous.

The description above is of a generic neuron. There is one major exception to this description. There are a number of categories of neuron which lack axons, collectively these neurons are known as *interneurons*. All interneurons lie entirely within a centre and they appear to form communication networks connecting the dendrites of large numbers of other neurons in a centre.

As previously stated the brain is organised into *centres*, each of which performs a specific task or tasks. Detailed examination of centres has found that they only contain cells from a

few categories. In general a centre is organised such that its inputs are taken from the axons of neurons in other centres and its outputs are the axons of a subset of its own neurons. All communication between centres is therefore via axons, with axons which connect the same pair of centres usually forming axon bundles. This organisation implies that there is no direct interaction between the dendrites of neurons in different centres.

# 3 The Action and Electrotonic Potentials

At an early stage in neurobiology it was postulated that the neurons used an electrical signalling mechanism [2]. Since then, investigations have demonstrated that there are two types of signal, the action potential and the electrotonic potential. The action potential is a pulsed potential, whilst the electrotonic potential is continuous. These properties have lead to the action potential being equated to a digital signal. The electrotonic potential is then equated to an analog signal.

The majority of experiments in neurobiology have concentrated upon the action potential, which is relatively easy to observe in accessible axons. Historically, this emphasis lead to a picture of neural information processing in which dendrites simply aggregated inputs from the axons of other cells. This aggregated input was then thought to trigger the axon attached to the dendrite. This picture formed the basis of the McCulloch and Pitts model of a neuron. Recently this emphasis has been changed. During the 1980s the technology became available which enabled studies of the electrotonic signals in dendrites[4]. It is the insight gained from these recent studies which suggests that a review of neurobiology may be fruitful.

## 3.1 The Electrotonic Potential

Electrotonic potentials are analog signals employed in the dendrites of neurons. In some neurons, the interneurons, they are the only signalling mechanism.

Historically the action potential was thought to be the only mechanism for inter-neuron communication. This picture changed with the discovery of electrotonically activated dendro-dendritic synapses. The only possible function of these synapses could be to communicate an electrotonic potential to another neuron[4]. The fact that inter-centre communication occurs via axons, and hence action potentials, implies that an electrotonic potential can only be communicated to another neuron within the same centre. Despite this apparent limitation the importance of the electrotonic potential should not be underestimated. In key regions, such as the retina, the electrotonic potentials would appear to be the dominant mechanism for information processing and communication.

[2] As early as 1868 Helmholtz was comparing neurons to a telegraph system[3]

### 3.2 The Action Potential

Although the action potential behaviour appears to be very sophisticated, and might be expected to occur only in higher animals, it is widespread, occurring in vertebrates and invertebrates.

An action potential is initiated in the axon hillock region of the soma. The shape of the axon hillock is thought to direct the action potential into the axon. To initiate an action potential the electrotonic potential of the soma has to increase above a *threshold potential*. Once the threshold is crossed the action potential is initiated and propagates down the axon. After an action potential has traversed a section of membrane there is a period during which another action potential cannot pass. There are two consequences of this effect: Firstly, the axon hillock is prevented from initiating another action potential during this *absolutely refractory period*. Secondly, an action potential can only propagate in one direction. To understand the latter effect consider a snapshot of an action potential. The field across the edges of the pulse will force the pulse to spread in both directions. The absolute refractory period means that the consequences of this spread depends upon the direction of propagation. In the forward direction the potential at a point just in front of the pulse will increase across the threshold potential. The potential at this point will therefore increase to its maximum value. On the contrary, in the backward direction the absolute refractory period means that the positive feedback mechanism which would increase the membrane potential to its maximum value is unavailable. Leakage currents will therefore cause the membrane potential to decrease. The result is uni-directional propagation.

This absolutely refractory period is followed by the *relatively refractory period*. During this period the threshold for action potential initiation gradually decreases towards its normal value. This effect means that at the axon hillock the time between successive action potentials depends upon the somatic electrotonic potential, with higher values leading to more rapid pulse initiation. The result is that the axon hillock can frequency encode the electrotonic potential of the soma.

The threshold potential for action potential initiation is not time independent. Variations in the membrane characteristics of the soma, due to its previous history, cause changes in the threshold. There are two phenomena in neurons which appear to be threshold variations, *accommodation* and *post-inhibitory rebound* (PIRB). Accommodation occurs when the threshold for firing is increased due to the accumulation of sub-threshold stimulus. PIRB is the ability of a neuron to fire action potentials as a response to a subthreshold input after a lengthy quiescent period. The fact that the action potential initiation threshold depends upon the previous history of the membrane has inspired models in which action potential initiation forms the basis of computation. Detailed studies have indicated that the action potential together with the soma thresholding action form a sufficient basis for Boolean logic.

## 4 Ionic Conductance Mechanisms

In early experiments on squid axons Hodgkin and Huxley identified a three ion mechanism which sustained signal propagation. The three ions involved were; $K^+$, $Na^+$ and $Cl^-$.

The $K^+$ has been found to be concentrated inside the neuron, whilst both the other ion types are concentrated outside the neuron[3]. The spatial separation of ions means there are two components to the ion current through the cell membrane. The concentration gradients cause *diffusion currents*, whilst, the electric field due to the charge separation causes drift currents. Each ion is characterised by the potential across the cell which balances the two components of its ion current. This *reversal* potential has been found to vary between cell types, examples, for the widely studied squid axon are: $K^+ = -92$mV, $Na^+ = 55$mV and $Cl^- = -65$mV[8].

The potential when the cell is in equilibrium is known as the *resting* potential. This can vary along the length of a single neuron. Together with the reversal potential each ion is characterised by the ease with which it can pass through the cell membrane. This is usually specified as an ionic *conductance*. If one of the ionic conductances is much larger than that of the other ions, the cells resting potential will be close to the reversal potential of that particular ion. In most cells the $K^+$ conductance is the largest and the cell is at equilibrium near to the $K^+$ reversal potential which is negative.

If the ion conductances vary in time the resting potential of a neuron will also become time dependent. In general an increase in the $K^+$ ion conductance will move the resting potential further from zero, and the cell is said to be hyperpolarised or inhibited. A cell whose potential moves closer to zero is described as depolarised or excited, an effect which arises from a non-specific increase in the conductances of the positive ions[4].

These simple considerations demonstrate how the voltage of a cell is affected by ionic conductances through the cell membrane. These membrane ionic conductances depend upon *micro-channels* formed by long chemical penetrating the membrane. The detailed behaviour of a membrane therefore depends upon the types of ion channels which it contains. Some channels have been found which react to changing voltages across the membrane by opening or closing. The mechanism for channel opening depends upon molecules forming the channel. These molecules appear to operate in groups of three with the penetration process driven by clusters of charge, usually $6e$, interacting with the voltage across the membrane. This use of clustered charge has the advantage that the penetration process is characterised by energies 6 times the energy which would correspond to a single charge. The clustered charge therefore amplifies the energy difference which drives the channel opening mechanism. This effect can be used to either reduce the driving fields, hence avoiding high field effects like dielectric breakdown, or increase the speed of response for a fixed driving potential. Detailed experiments have shown that the conductances have an exponential dependence on voltage which saturates after a few decades increase in ion current. In the exponential region the typical voltage dependences are 3.9mV/e-fold for $Na^+$ and 4.8mV/e-fold for $K^+$ [8]. These figures are equivalent to 9.0 mV/decade for $Na^+$ and 11.1mV/decade for $K^+$ [4].

The effect of opening an ion channel depends upon the ion species involved:

- $Cl^-$ is in greater quantity outside the cell. If the resting potential is negative the fact

[3]This is common to all cells in the body and is not a special adaption of neurons to enable signal propagation.

[4]This behaviour is far superior to a silicon MOSFET, which has a typical subthreshold slope of 100mV/decade and a best achievable of 60mV/decade in specialist silicon-on-insulator technologies.

that $Cl^-$ is a negative ion means that the potential gradient across the cell can balance the diffusion gradient. Therefore the opening of chlorine channels whilst the neuron is at rest has little or no effect. However if the potential of the cell deviates from rest the electric field will no longer balance the diffusion gradient. The chlorine ions will flow to try to restore the membrane potential to its resting potential, performing a shunting action[9]. This shunting is not effective enough to totally isolate sections of the neuron, it simply reduces the magnitude of the signal propagating past the point at which the $Cl^-$ channels are opened.

- As previously stated, the opening the $K^+$ channels hyperpolarises the cell. In general the resting potential is already close to the $K^+$ reversal potential, the maximum amount of membrane hyperpolarisation is restricted, a typical upper limit is 10mV.

- Opening $Na^+$ channels moves the resting potential closer to zero, i.e. more positive, the cell is said to be de-polarised. Depolarisation of the cell causes synaptic activity and stimulates action potentials has lead depolarisation to be referred to as excitation. It is the state of depolarisation of a cell which is communicated to other cells via synapses or action potentials.

The fact that ionic conductances underly action potentials was first demonstrated by Hodgkin and Huxley. They devised equations, based upon ion conductances, which gave a good quantitative description of a single action potential.

The bistable nature of the action potential, used to enable long distance transmission, requires a positive feedback mechanism. The positive feedback is due to a voltage dependent $Na^+$ conductance. This mechanism relies upon a $Na^+$ current determined by the product of two factors; the conductance and the driving potential $(V - E_{Na})$, where $E_{Na}$ is the $Na^+$ resting potential. As the cell depolarises the driving potential decreases, whilst the conductance increases. The threshold voltage for action potential initiation is the voltage at which the rate of increase of the conductance is greater than the rate of decrease of the driving potential. Once the voltage is greater than the threshold it continues to increase until the driving potential, $(V - E_{Na})$, is zero. Once the $Na^+$ resting potential is reached the $Na^+$ current mechanism automatically de-activates. This de-activation is thought to arise from changes in the molecules which form the channels. It has been postulated that these molecules contain structures which eventually move to block the channel[10]. The de-activation mechanism is therefore separate from the activation mechanism. The cell is returned to its normal resting potential by the action of a slow voltage dependent $K^+$ conductance. Once the membrane potential has returned to its usual resting value the molecules which form the $Na^+$ channels return to the membrane surface.

During the period, just after the passage of an action potential, in which the ion channels are blocked, the channels cannot contribute to another action potential. This is the *the absolutely refractory period* during which action potential propagation is blocked. During the period in which the ionic concentrations, perturbed by the passage of the action potential, are returning to their usual values the membrane is more difficult to stimulation, this period is referred to as the *relative refractory period*[5].

[5]The uses of these refractory periods by the system are discussed section 3.2

The Hodgkin and Huxley model explains the phenomena behind action potentials, however it does not include all the underlying physics and is therefore suspect. The simplest example of this, is the failure of the equations to model the timing between the generation of two action potentials. A failure which appears to arise from the fact that the model includes a long time tail to the $K^+$ conductance which tended to hyperpolarise the cell. Despite its inadequacies the Hodgkin and Huxley model is sufficiently correct to form the basis of other work[11]. Kernell adapted the model to allow for exponential decay of the conductance, and adaptive behaviour. Inspired by Hebb's ideas on learning the model adapts behaviour by including a conductance which depended upon successive output spikes. The generation of an action potential caused the conductance to be incremented.

Since the early work, which identified the three ion system, $Ca^{2+}$ has been identified as an important ion, especially in the dendrites[11]. The full implications of the presence of $Ca^{2+}$ and its exact function has yet to be determined. However the possible importance of $Ca^{2+}$ is emphasised by the fact that this failure to understand the role of $Ca^{2+}$ is at least partly due to the complexity and scope of the phenomena in which it is involved. The key features would appear to be:

- $Ca^{2+}$ is used in both electrical conduction and metabolic processes. This link is particularly important in developing neurons. In these neurons the $Ca^{2+}$ ions form the only electrical signalling mechanism available. The interaction of the $Ca^{2+}$ present due to electrical stimulation and the cells metabolism may play a key role in the growth and movement of the developing neuron, which determines the final location and connectivity of the mature neuron[12]. In mature neurons $Ca^{2+}$ could provide the link between the electric activity and the structure of the neurons which is thought to be important in long term memory.

- $Ca^{2+}$ ions can have a voltage dependent conductance similar to that of $Na^+$, a vestige of the mechanism in developing cells. In the majority of neurons the $Ca^{2+}$ mechanism is replaced by the $Na^+$ mechanism, however in some neurons the action potential behaviour can be based upon $Ca^{2+}$ currents rather than $Na^+$ currents. In extreme examples the $Ca^{2+}$ and $Na^+$ mechanisms can occur simultaneously in a single neuron. The most striking example of this occurs in the Purkinje cell in the cerebellum, which employs a $Ca^{2+}$ mechanism in its dendrites and a $Na^+$ mechanism in its axon. These action potentials interact to produce complex signals in the axon[3], an interesting phenomenon, whose exact function is unclear.

- In some neurons a change in the extracellular $Ca^{2+}$ levels is equivalent to a change in the resting potential. A five-fold increase in the amount of extracellular $Ca^{2+}$ is equivalent to a $10-15$mV shift in the resting potential. This appears to be important in changes which occur to the soma to alter the threshold for action potential initiation.

- The fact that the threshold for action potential initiation depends upon the previous history of the membrane, for example accommodation, suggests that action potential initiation could be a basis for computation. The range of responses observed when a constant input current is applied is thought to be due to interactions between a $Ca^{2+}$ dependent $K^+$ current, a $Ca^{2+}$ current and adaption by the first part of the axon. The two currents are thought to interact to determine the frequency of action potential initiation for a constant input current.

- The channels for $Ca^{2+}$ ions in the cellular membranes may be the only channels to be under direct metabolic control.

All the ion conductance mechanisms discussed previously are based upon a neurons ability to maintain a concentration gradient. The $Na^+$ and $K^+$ ion concentration gradients are maintained by the active pumping of ions across the membrane. This function is performed by special structures in the cell membrane, the *ion pumps*. The pumping process consumes ATP, a general cell energy source, and is only activated when the distribution of ions deviates from the resting distribution. The consumption of ATP in the pumping process means that the pumps form an important link between the metabolic system and information processing. During one pump cycle 3 $Na^+$ ions are moved into the cell and 2 $K^+$ ions are moved out. This means that the pump acts to charge the membrane and a sustained period of pumping will act to hyperpolarise the membrane. To maintain the concentration differences the density of pumps in a membrane has been found to be approximately the same as the density of ion channels. The $Ca^{2+}$ content has been found to be controlled by the coupled transport of $Na^+$ and $Ca^{2+}$ through the membrane. The movement of 3 $Na^+$ ions along the potential gradient into the cell provides the energy source for the expulsion of one $Ca^{2+}$ ion against its concentration gradient. In contrast to the other ions the $Cl^-$ ion concentration does not appear to be controlled actively. By maintaining the concentration gradients the ion pumps and the coupled transport mechanism are analogous to electrical power supplies in electronics.

This section represents only the briefest account of the current knowledge of the mechanisms underlying variations in ionic conductances across neural membranes. The ionic conductances make available a wide range of behaviours for potential application to information processing. We now go on to consider how the ion conductances translate into the behaviour of components of the neurons.

# 5 Membranes and Synapses

In this section the behaviour of component parts of neurons are discussed. Since this document is concerned with the information processing activities of neurons, discussion will be limited to two parts of the neuron; the neuron membrane, which supports signal propagation, and the neuron synapses, which initiate signals.

## 5.1 Membrane Characteristics

By supporting a potential difference, the membrane is *the* part of the neuron which enables signal propagation. From the previous discussion of the ionic conductance mechanisms it is clear that the properties of a patch of membrane depend upon the types of ion channels which it contains.

The simplest membranes to model and understand are *passive* membranes, which contain no voltage dependent ion channels. There is a high resistance to current flow across these membranes and they can be considered to be dielectrics. If a voltage source is applied

to a point on a passive membrane current will flow along the neuron, through a resistance per unit length $R_i$, to the neighbourhood of the source. This current flow will attempt to charge the membrane capacitance to the applied voltage. However, the finite membrane resistance means that some of the current leaks away. The result is a voltage which decreases as the distance from the source increases. In essence a length of neuron with a passive membrane behaves like a conducting cable with a finite leakage through its insulating cover. This means that the the work of Lord Kelvin and Heavisides, undertaken in the $19^{th}$ century for application to telegraph wires and the first transatlantic cable, can be applied to neurons[3].

A membrane can be characterised by either one, of two sets of three parameters:

- The first set of parameters are identifiable properties of the membrane. These are $r_i$, the longitudinal resistance ($\Omega cm$), $r_m$, the membrane resistance per unit length ($\Omega/cm$) and $c_m$, the membrane capacitance per unit length ($\mu F/cm$).
- The second set of parameters are derived electrical characteristics. These are a characteristic decay length, $\lambda$, the input resistance, $r_{input}$ and the RC time constant, $\tau$[3].

These two sets of parameters are related, the relationships being:

$$\lambda = \left(\frac{r_m}{r_i}\right)^{1/2} \tag{1}$$

$$r_{input} = 0.5(r_m r_i)^{1/2} \tag{2}$$

$$\tau = r_m c_m \tag{3}$$

The existence of a characteristic decay length is a consequence of the current leakage across the membrane. This means that equilibrium is established with a voltage which decays as the longitudinal distance from the voltage source, $d$, increases. In the simplest case the voltage decays exponentially and is given by

$$V(d) = V \exp\left(-\frac{d}{\lambda}\right) \tag{4}$$

where $V$ is the voltage applied at the source.

In general the overall length of a dendrite is approximately equal to its characteristic length[11]. Neurons which use electrotonic potentials over extended distances have adapted by changing their membrane characteristics to have increased characteristic lengths. The approximate equality between the actual length and the characteristic length means that the potential distribution in dendrites depends upon the boundary conditions representing the dendrite terminals and the soma. For example, if a terminal boundary is equivalent to a normal patch of membrane, the lack of a leakage path beyond the termination boosts the potential at the terminal. The result is that the potential between the source and the terminal is higher than expected. Alternatively the potential change caused by a source can be reduced. This occurs when the leakage current across the membrane is increased thereby

decreasing the local characteristic decay length, $\lambda$, This increased leakage current can arise when any local $Cl^-$ conductance channels are opened. This short circuits the membrane voltage and the voltage decays more rapidly than expected between the source and the sink.

Another point which has emerged is that the overall length of an axon is usually greater than its characteristic length. The relationships between the characteristic lengths and the total lengths of axons and dendrites are therefore different. This may be *the* significant difference between axons and dendrites. This viewpoint may be supported by the fact that short neurons exhibit only electrotonic potential with no action potential behaviour. If valid, this argument would suggest that axons have been forced adopt pulse communication because of their overall length.

The advantages of action potentials in myelinated axons can now be appreciated. A myelinated axon has a covering of insulation with periodic gaps, the nodes of Ranvier. This insulation has two effects: it reduces the membrane capacitance and prevents leakage of current. The result is that any current passing one node will charge the reduced capacitance of the following length with no losses. The overall result is a more rapid propagation of the action potential when compared to an unmyelinated axons. If the $Na^+$ current across the membrane was entirely prevented the mechanism underlying the action potential would be blocked. The nodes are therefore placed such that the voltage change at one node can force the voltage at the next node above threshold. Once the nodal voltage crosses the threshold voltage the $Na^+$ mechanism drives it to the $Na^+$ resting potential, thus restoring the signal level. The result is the rapid propagation of an unattenuated signal.

Another advantage of the action potential is that the positive feedback, due to $Na^+$ conductance mechanism, ensures that the soma can initiate an action potential independent of the overall length of the axon. This is a requirement placed on the system by the fact that the final length of the axon is not predetermined but is the result of growth during development. The action potential mechanism means that the soma is capable of initiating an action potential in any length of axon.

The different relationships between the characteristic length and the overall length of axons and dendrites may suggest that the action potential mechanism is used for communication. This view appears to be taken by Koch and Poggio[6] despite the fact that action potential initiation could form a basis for Boolean logic.

Now reconsider the expression for the input resistance of a section of neuron shows that it varies as the neuron diameter raised to the power of 3/2[3]. This means that the condition

$$d_o^{3/2} = \sum_1^n d_i^{3/2}$$

applied to the junction between $n$ input fibres, diameter $d_i$, and an output fibre, diameter $d_o$ is equivalent to impedance matching.

Since this condition can be applied to axons and dendrites, the impedance matching condition applies to both. A close examination of dendrite junctions suggests that this relationship holds for most junctions, i.e. most junctions are impedance matched. In axons the impedance matching condition can be applied to the bifurcation of an axon. It has been suggested that the membrane characteristics and diameters of the daughter branches could

be altered to prevent the action potential from invading one or other of the daughters. This behaviour could be controlled by the the geometric ratio, GR, defined as the ratio of the two sides of the impedance matching condition,

$$GR = \frac{\sum_1^n d_i^{3/2}}{d_o^{3/2}}$$

If $GR = 1$, the condition for impedance matching, the action potential propagates through the branch point unhindered. In the regime $1 < GR < 10$ the bifurcation causes delay. For $GR > 10$ propagation fails beyond the bifurcation point. The geometric ratio is fixed and can never be used to differentially route data. However, there is evidence from invertebrates that different frequencies can be used to route the action potential down different daughter branches[6]. This behaviour requires daughter branches with different frequency dependent membrane characteristics and is the only information processing function that has been observed in axons.

Now consider the dynamics of the electrotonic potential. Slow variations can be approximated by the steady state. However, a more detailed treatment of temporal variations needs to include the effects of charging the membrane capacitance and injection of current via voltage dependent conduction channels. Rall[11] has applied cable theory to a section of neuron. He found that the spatio-temporal evolution of the electrotonic potential in the section of neuron can be represented by the equation:

$$\frac{1}{2\pi r(r_i + r_e)} \frac{\partial^2 E}{\partial x^2} = c_m \frac{\partial E}{\partial t} + GE + \sum g_i(E - E_i) + I \qquad (5)$$

In this equation E is the electrotonic equation in a neuron with a radius, r. The first term,$c_m \frac{\partial E}{\partial t}$, represents the charging of the membrane capacitance. $GE$ represents the resting conductance across the membrane. To this a contribution from the active conductances per unit area $g_i$, each of which has a characteristic equilibrium potential, $E_i$, is added. The final term, I, represents the action of any external current source, which could be a synapse or an experimental probe.

Applying this equation to passive membranes, Rall was able to prove that if all the dendritic junctions in a tree obeyed the impedance matching condition, the entire tree can be represented by a single cable. This equivalence has been used to dramatically simplify the study of dendritic trees, in which each dendrite can be considered to be a delay line.

As in electrical cables the necessity to charge the membrane capacitance via the longitudinal resistance means that a length of dendrite acts as a low pass filter. This restricts the signal bandwidth to be the inverse of the RC time constant. Experimentally the time constants of mammalian neurons have been found to be in the range 5 – 20ms[11] which corresponds to bandwidths in the range 50 – 200 Hz.

A length of neuron will also degrade a signal by adding noise. Fortunately, the restricted signal bandwidth acts to limit the added noise power per unit length, this added noise power per unit length per Hz of bandwidth at the angular frequency $\omega$, $S_V(\omega)$, is given by

$$S_V(\omega) = \frac{4kTr_i}{1 + (\omega c_m r_i)^2} \qquad (6)$$

This noise level may be important in establishing the signal dynamic range which a membrane can support and the distance over which an input has a measurable effect.

Rall and co-workers have also applied the cable equation to model the time dependence of electrotonic signals. This work has demonstrated the relevance of the spatial location of a synapse. First consider the effect upon the detected signal of increasing the distance between the input position and the measurement position, usually the soma. If the two points are close, then the time variations are rapid and the voltage excursion is large. As the distance between the points increases then the maximum excursion and the rate of change of the signal both decrease. Similarly, Rall has studied the affect of varying the relative positions of excitatory and inhibitory inputs[4]. To do this Rall used a five node model, with the excitatory signal applied to the centre node, node 3, and the result monitored at node 5. He then compared the result of putting a permanent inhibition on nodes 1, 3 and 5 in turn to the result with no inhibition. The results were:

- If the inhibition is on the far node, node 1, then the impact is a slightly increased rate of decay after the peak has passed.
- With the inhibition at node 3, the site of excitation, then the result is a general reduction in the magnitude of the signal by a factor of approximately two.
- If the inhibition is at the node were the signal is monitored then the effect is again an overall reduction but greater than with the inhibition at node 3.

Since these results are very model dependent they can only be considered to be qualitative. However, this work demonstrates that the response is **not** a simple linear addition of the responses caused by the inhibition and the excitation. The model also indicates that the relative positions of the inhibitory and excitatory synapses are the critical factor in determining the behaviour of a neuron.

The cable equation is most easily applied to passive membranes. Unfortunately passive membranes are very rare and it is now thought that most neurons exhibit significant and localised active membrane conductances. The voltage dependent conductance channels mean that an active membrane can respond to voltage changes and may add to or subtract from the signal (the most extreme form of active membrane is one which contains a positive feedback mechanism which can generate an action potential). In general there is also a behaviour between these two extremes which Koch and Poggio refer to as quasi-active[6]. This behaviour is based upon ion conductance channels which can be modelled as inductors. The addition of this inductor to the existing RC equivalent circuit gives rise to the possibility of the membrane acting like a band pass filter.

This short discussion suggests that the exact behaviour of membranes is varied and complex, depending upon the distribution of ionic conduction channels within the membrane. It would appear that the behaviour of membranes has yet to be fully understood, with the only membranes which we understand in any detail being passive membranes.

## 5.2 Synapses

In general neurons communicate via sites of special adaptation, the synapses. Detailed studies have shown that there are two major types of synapses, the *gap junction* and the *chemical synapse*. A gap junction is an electrical contact between two neighbouring cells whilst a chemical synapse, the predominant type of synapse, employs chemical signals.

### 5.2.1 Gap Junctions

Structurally, a gap junction is a region in which two neurons are separated by $2-4$nm over an area with a diameter in the range $0.1-10\mu$m. The membranes on either side of the gap junction have been found to contain ion channels with relatively large conductances, of the order of 120pS. The resulting low resistance to current flow means that a change in the presynaptic potential is transmitted to the post-synaptic neuron with little attenuation or delay. The functioning of a gap junction depends upon voltage dependent ion channels which give rise to voltage dependant currents across the membrane. It has been found that the current voltage characteristics are linear in the range 25mV either side of the equilibrium potential. In general the input impedances of the two sides of the junction are different, with a sufficiently large difference giving rise to a strongly rectifying junction. The behaviour of a specific junction is not fixed, the ion channels can be gated by a variety of factors including intracellular free $Ca^{2+}$ and pH[12]. The result is that the transfer characteristics of a junction can be sensitive to the intracellular pH, the voltage across the junction and local inhibitory synapses.

The exact function of gap junctions is unknown. Only a few sites containing gap junctions have been identified. In some instances the gap junctions are associated with chemical synapses between other neurons. The geometry of these connections are usually such that the gap junctions are at the end of a dendrite branch with a chemical synapse from a third neuron positioned to short out the action of the gap junction. The exact function of this type of structure is unknown. In more specialist applications it has been suggested that the use of gap junctions in the vertebrate retina may help be improve the signal to noise ratio of the detected signal[6]. Whilst, some postulated applications are based upon the fact that, unlike chemical synapses, gap junctions introduce zero transfer delay. One such application is the possibility that gap junctions play an important role in co-ordinating the action of large numbers of neurons in specialist tasks, such as generating the heart pace-maker signal.

### 5.2.2 Chemical Synapses

The name chemical synapse arises because these synapses employ chemicals to communication between neurons. This chemical signal, the *neurotransmitter*, is produced in small structures, vesicles, in one neuron in each neuron pair forming a particular synapse. This observation implies that a signal can only be initiated by one of the cells, any information transfer is therefore uni-directional. The cell which releases the neurotransmitter is termed the pre-synaptic cell. The neurotransmitter is received by the post-synaptic cell. The impact upon the post-synaptic cell of the release of the neurotransmitter depends upon the receptor chemicals on its surface which interact with the neurotransmitter.

Chemical synapses are usually categorised by the parts of the pre-synaptic and post-synaptic neurons which are connected. The three general categories are:

- The axo-dendritic synapses, the first synapses to be discovered, and, until the 1960s the only synapses which were thought to exist [6].
- Dendro-dendritic synapses were first discovered in 1965 in the olfactory bulb[5]. Attempts to understand their behaviour is complicated by the fact that they tend to form reciprocal pairs or small groups and their exact function is unknown[11]. The first impact of their discovery was to explain the existence of the interneurons; neurons without axons and therefore no axo-dendritic synapses. Shepherd[5] has argued that in general in small groups these synapses are a logical and economical way to organise interactions.
- The third type of synapse, the axon-axon synapse, is usually found in conjunction with axo-dendritic synapses. The configuration is usually such that an axo-dendritic synapse between a pair of a cells is directly influenced by a third cell. The influence takes the form of a synapse from the axon of the third cell onto the axon of the pre-synaptic cell close to the axo-dendritic synapse. This axon-axon synapse can have a considerable effect on the axo-dendritic synapse, however, timing is critical since any effect only lasts for a few milliseconds.

As well as categorising synapses by there position studies have identified two general types of synapse distinguished by their general structural characteristics[12]:

1. Type I synapses have a large area, $1 - 2\mu$m diameter, with a synaptic cleft of approximately 30nm. They typically contain large, $30 - 60$nm spherical vesicles.
2. Type II synapses have a smaller area, typically with a diameter less than $1\mu$m, and a narrower cleft, 20nm. The vesicles tend to be small, $10 - 30$nm, and elliptic.

The function of a chemical synapse is to release neuro-transmitters from the vesicles in the pre-synaptic cell in response to its depolarisation. The neurotransmitters interact with the post-synaptic membrane by binding to receptor chemicals to open molecular ion channels. These channels allow ions to pass through the membrane, hence changing the post-synaptic cell potential. The result is that the pre-synaptic depolarisation is communicated to the post-synaptic cell.

This communication mechanism means that the function of a synapse dependents upon the neurotransmitter-receptor pair of chemicals. To make things even more complex each receptor cell may also possess a range of different receptor chemicals which are triggered by the same neurotransmitter to perform different tasks. One important consequence of the fact that the synaptic function is determined by both the pre-synaptic and post-synaptic cells is to restrict the applicability of Dales Principle. In its original form Dales principle suggests that the metabolic unity of a cell suggests that all its synapses would release the

[6] These synapses were the only synapses known to both McCulloch and Pitts, when they invented their thresholding neuron, and Rosenblatt when he invented the perceptron.

same neurotransmitter. Neurobiologists have found it convenient to study accessible parts of neurons, and then use Dales principle to extrapolate their results to the other parts of the neuron. In the past they have been tempted to suggest that a neuron under investigation is either excitatory or inhibitory depending upon the neurotransmitter. As Shepherd[4] has noted such statements are an extrapolation of Dales principle far beyond its original statement. There appears to be no evidence that pairs of chemicals which have the same effect upon the postsynaptic potential correspond to the same vesicle structure. This implies that any attempt to suggest that the two types of synapse, described above, correspond to inhibitory and excitatory synapses is invalid.

There have been up to 50 neurotransmitters identified. This apparent over provision of neurotransmitters could be the result of chemical synapses evolving from a hormonal message system as a more efficient alternative[13]. It is this evolution which would then cause the apparently unnecessary redundancy in the number of neurotransmitters and receptors observed in nervous systems.

The relationship between the presynaptic and postsynaptic potentials is usually thought to be either almost linear or sigmoidal. The roll-off in the sigmoidal response could be caused by two mechanisms, the saturation of the receptor chemicals, or, the fact that the postsynaptic potential is approaching the reversal potential of the ion carrying the membrane driving current. Any synaptic saturation limits the dynamic range of the synapse. An example of an observed dynamic range is that of insect monopolar cells, with a dynamic range of $10^1 - 10^2$ compared to the $10^5$ range of the pre-synaptic photoreceptor cell.

As well as the dynamic range, another critical parameter of a synapse is its sensitivity or dynamic gain $\partial V_{post}/\partial V_{pre}$. The conditions for maximum dynamic gain identify the most effective operating point of a synapse. Koch and Poggio [6] quote dynamic gains in the range $0.3 - 34$ and have suggested that there are two major categories of synapses. The first category has an operating point with a presynaptic polarisation close to zero and a large dynamic gain. Whilst the second category operates with significant presynaptic polarisations ($60 - 80$mV) with a small dynamic gain, $\approx 1$. (The widely studied squid axon synapses belong to this second category.) Koch and Poggio think that these characteristics indicate that the first category communicates analog signals whilst the second category translates a digital signal to an analog signal [7]

In both dendrites and axons the amount of neurotransmitter released depends upon the degree of depolarisation of the pre-synaptic membrane. In axons it is the time integrated signal which causes the depolarisation[4]. Experiments with the synapses of squid axons suggest that the depolarisation acts to create an influx of $Ca^{2+}$ into the pre-synaptic cell, with the opening of these channels being the rate limiting process. This $Ca^{2+}$ influx initiates a chain reaction, which results in vesicles fusing with the membrane of the synapse, precipitating the release of the neurotransmitter from the vesicle[12].

The fusing of a vesicle with the pre-synaptic cell wall releases a "quantum" of neuro-

[7] It might be tempting to identify these two categories with the structural categories at the beginning of this section. Unfortunately, I was not able to establish this link. I could not find a reference which discussed both topics. The only hint I found was that the type I synapses tended to be axodendritic synapses[7], in the same reference type II synapses are listed as axosomatic. However this author falls into the trap of trying to identify the two types with inhibitory and excitatory behaviour.

transmitter. The number of molecules in each quantum can vary, from 3 to 1000, depending upon the synapse and its application. In neuromuscular junctions the number of molecules in a quantum is large, indicating a requirement to convey a clear measure of the presynaptic potential. On the contrary, neurons in the brain with large numbers of inputs appear to have small numbers of molecules in a quantum[3]. Within each synapse the size of a quantum is relatively constant, with the probability of a quantum containing a specific number of molecules determined by the Binomial distribution.

For a fixed quantum size the different levels of pre-synaptic depolarisation are communicated by causing the release of different numbers of quanta, with a typical signal corresponding to approximately 100 quanta. Experiments with reduced amounts of $Ca^{2+}$ in the synaptic cleft have shown that a single quantum causes a $0.4 - 1.0$mV change in the postsynaptic potential. Other experiments based upon noise analysis have shown that the current due to a single quantum of neurotransmitter can vary by 1nA in 120nA, suggesting that approximately 100 ion channels are opened by a quantum. (This result is consistent with direct estimates of channel conductances in other synapses which were found to be $20-30$pS.) Noise analysis has also shown that the time a channel remains open is distributed exponentially, with a mean of the order of 1ms. The channel closure mechanism was found to be accelerated by de-polarisation and slowed by hyper-polarisation.

In general, it is now believed that long term changes in the synaptic efficiency are due to changes in the number of neurotransmitter molecules in a quantum[3]. However there are instances in which the number of quanta released in response to a fixed presynaptic potential can vary. If a synapse is stimulated by a stream of identical pulses the postsynaptic response to each one increases. This phenomena, facilitation, is caused by an increase in the number of quanta released by the same stimulus, and, is thought to be caused by a build up in the level of $Ca^{2+}$ in the pre-synaptic cell. Any facilitation which occurs has been found to persist over periods of the order of 100ms. There are similar long term effects known as augmentation, lasting seconds, and potentiation which can persist from seconds to hours. These latter two phenomena are thought to be linked to the release of $Ca^{2+}$ from $Ca^{2+}$ supplies within the pre-synaptic cell.

The synaptic cleft has been found to contain enzymes which attack the neurotransmitters, thus performing the important function of restricting neurotransmitter action, both spatially and temporally. By restricting the spatial range of the neurotransmitter, the enzyme ensures that a quantum only affects the patch of postsynaptic membrane nearest its point of release[3]. This is important in ensuring that each quantum acts independently by preventing the local saturation of the receptor chemical concentration. Temporally the enzyme prevents the build up of neurotransmitter in the synapse, ensuring that the synapse is not saturated by its previous history.

The type of synapse described above operated by increasing the ionic conductance of a membrane. There are also synapses which operate by decreasing the membrane ionic conductance. These synapses therefore act as normally open shunting inhibitions which are closed when the synapse activates. Thus, activation of one of these synapses will increase the range of influence of other local synapses. Similarly by decreasing the local leakage current, synaptic activation increases the period over which temporal integration of other inputs occurs. Hence, the result of synaptic activation of this type of synapse is to extend the spatial and temporal influence of other local synapses[12].

So far we have only described junctions between two neurons. It has been discovered that the release of neurotransmitters into a synaptic cleft can be inhibited by input into the pre-synaptic cell from a third neuron. This inhibition acts by decreasing the amount of neurotransmitter released in response to a presynaptic potential. It has been found that to be effective the pre-synaptic inhibition has to be critically timed, with an effect which only lasts for a few milliseconds.

### 5.2.3 Dendritic Spines

So far there has been an implicit assumption that any synapses are formed directly upon the main branches of the cell. This is not universally true. Some synapses are connected to short side branches from the neurons, known as spines. This leads to the classification of neurons in the nervous system as either spinous or aspinous. In the former case the majority of the excitatory inputs are localized on the end of short spines, and not directly on the dendrite. In some centres, for example the cerebellum, all the synapses are on spines[3].

The exact function of spines is yet to be clarified. The suggested functions include:

- Highly specific interaction between groups of inputs. Between 5% and 15% of cortical spines would appear to possess both inhibitory and excitatory synapses. Normally the activation of an inhibitory synapse on a dendrite affects all inputs further from the soma along the dendrite. By contrast inhibition near a spinal neck will simple affect the synapses on that particular synaptic bulb. An extension of this principle would suggest that the dendritic bulb can be considered to be a local "circuit", especially if the synapses allow two way communication between pairs of neurons.
- The strength of the input from a synapse is thought to depend strongly upon the spine geometry, even with a passive spinal membrane. If the spinal membrane is active, and there are sufficient synapses connecting to the spinal bulb to generate an action potential, the spinal neck may be able to amplify the action potential. Thus with an active membrane the postsynaptic potential can be amplified, with a passive membrane it can only be attenuated[6].

It is the latter mechanism, suggesting that spinal geometry may play an important role in memory, which has excited the contemporary interest in spines. It would appear that there have been two mechanisms suggested by which the spinal geometry may change with time: First, the adaptability of the spines may arise from the behaviour of a unique organelle located in the neck of the spine. The exact mechanism by which this organelle could influences the spinal shape not known. The key to the possible mechanism is that the organelle appears to be a $Ca^{2+}$ sequester, suggesting the involvement of $Ca^{2+}$. A combination of this mechanism with a voltage dependent $Ca^{2+}$ ion conductivity, would allow electrical stimulation of adaptation, in line with a learning process based upon Hebbs ideas. Secondly, on a shorter timescale it has also been postulated that the presence of contractile proteins in the spinal neck could lead to adaptation within a fraction of a second. Both of these mechanisms appear to be speculative and may represent memory acting on different timescales.

# 6 Concluding Remarks

From the existence of centres with specific functions, the first lesson to emerge from any study of the brain may be that we should investigate dedicated parallel processors. This simply reinforces the current trend to dedicated chips and distributed parallel processing. If this were the only result of our study we would be disappointed. Fortunately, more important lessons may emerge from an examination of how centres operate.

The possibility that at least some of the information processing in the brain occurs via electrotonic potentials arises from three observations: First, synapses can communicate any electrotonic membrane depolarisation. Second, individual dendrites possess different synapses at which they form the pre-synaptic or post-synaptic partner, i.e. they have both inputs and outputs. Finally, possibly the most important point, the interaction of synapses via dendrites is a non-linear process.

This possibility has increased the perceived importance of synapses. The exact function of a neuron is now thought to be determined by its synaptic organisation, a view supported by several observations:

- Synaptic behaviour is complex and diverse.
- Neurons retain the ability to change their synaptic connectivity. A cells connectivity can change in response to the learning of a new skill or in response to the death of a neighbouring cell which exposes sites for potential synaptic connection[12].
- Work on Aplysia has shown that this invertebrate uses changes in synaptic efficiency to perform associative learning[14].
- Genetic disorders are caused by abnormal development of groups of neurons. The mature brain therefore possesses an incorrect connectivity[12].

In an extreme form this argument leads to the conclusion that the synapse is the building block of the nervous system[4].

If the functionality of synapses is fundamental to the operation of the brain, we need to consider the possibility of reproducing this functionality in artificial 'synapses'. Even in this short text, the complexity of the mechanisms employed in synapses is evident. It may prove especially difficult to invent compact, artificial equivalents of the sequences of chemical events in the release of neurotransmitters. Unfortunately, the real difficulties will arise when attempting to mimic the array of adaptation mechanisms available in the brain. These difficulties will only be aggravated by any requirement to maintain compactness to allow large connectivities. Although taxing, these difficulties can not be avoided, especially if we wish to exploit some of the promised advantages of artificial neural networks, such as fault tolerance.

Further consideration indicates that synapses may not be the only structural feature of neurons which we are forced to mimic. Although the synapses *may* be the single most important feature in the brain, the way they interact on a dendrite is controlled by the dendritic membrane between the synapses. We may be forced to mimic the variety of

membrane behaviours available within a neuron. Unfortunately, it would appear that the full range of behaviour available to membranes is not fully understood. This will hinder attempts to mimic membranes, to the possible detriment of the entire system.

If we are attempting to understand the brain, as inspiration in our search for solutions to our problems, the outstanding difficulty we face is the complexity of the mechanisms employed in the brain. ***Despite this there is still hope.*** In the past the same difficulty was faced by McCulloch and Pitts, who achieved considerable success with their formal model of a neuron. Since we now know that this model is based upon only one aspect of the behaviour of a neuron, the thresholding action of the soma when firing an action potential, its success is even more impressive. Despite the limited understanding of brain behaviour which inspired the formal neuron model, when wired in the correct manner even these simple neurons can be used as the building blocks from which any computer *could* be built. We now know more about the brain. This may lead us to devise better computational devices.

## Further Reading

Unless a specific piece of data was required attempts to refer to primary material was found to be a lengthy process, requiring very careful reading. The book by Shepherd[12] is useful as an introduction to neurobiology with a short section on each of the major systems of the brain. A reader interested in information processing in the brain should refer to the book by Kuffler, Nicholls and Martin[3] but especially recommended is the memo by Koch and Poggio[6].

Although not directly connected with the subject of this memo an historical perspective of neural networks is also useful. In this context parts of Arbib[2] and the prologue and epilogue of Minsky and Papert[1] are short, but, useful.

## Acknowledgements

I would like to thank Dr. D. Lowe for his assistance during the writing of this memo.

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Abstract

Over the past decade there has been increasing interest in neurologically inspired computational techniques. This interest arises from the concurrence of two factors; firstly, a growing list of interesting tasks for which serial digital computers are unsuitable, and secondly, information gained from the application of new techniques in neurobiology. This text is intended to provide an introduction to neurobiological terms for physical scientists and engineers, with some pointers to further reading. As an introduction little prior knowledge is assumed and the text begins with a short description of a generic neuron. This description is followed by more detailed discussions of those aspects of neurobiology of particular importance in information processing. The topics covered include the following. The electrical signals used to represent information. The ion conduction mechanisms employed by cells to support these signals. The behaviour of cell membranes and intercellular junctions. Finally a few comments on the immediate implications of even a superficial understanding of neurobiology are included. —RH